

ROBERT A. ALDRICH, M.D.

# Clinical Research

## PROCEEDINGS

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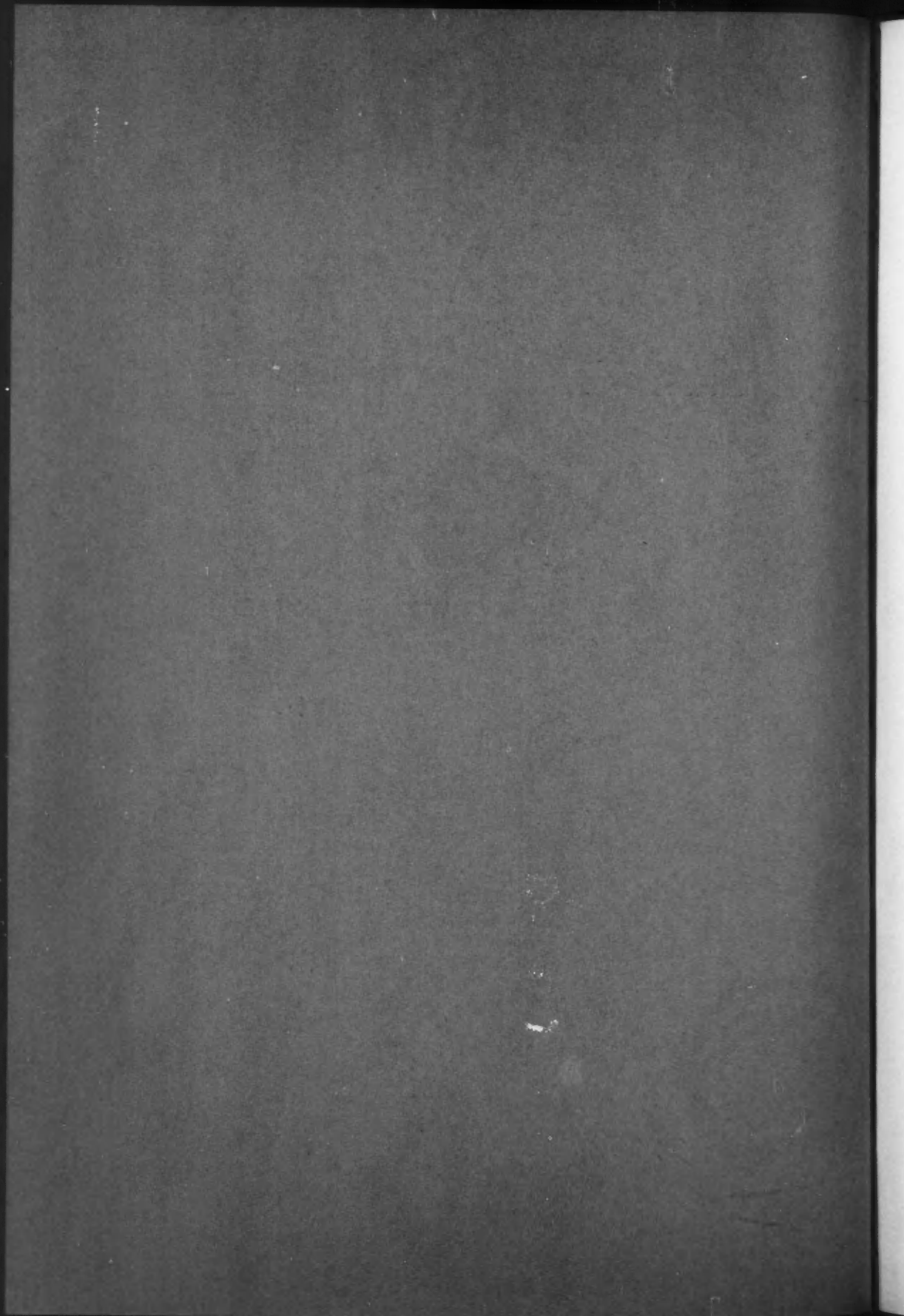
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### A Welcome Place\*

By William H. Beierwaltes

THE GREAT STRENGTH of the AMERICAN FEDERATION FOR CLINICAL RESEARCH is its successful endeavor to create a welcome place for every competent beginner in medical research to present a discussion of his work before an understanding and enthusiastic audience. The history of similar organizations, however, demonstrates that the task of continuing the promotion of this ideal will become increasingly difficult as our Society ages. It is my desire, therefore, to strengthen this endeavor by refreshing our memories of the plan of our organization with a brief narrative of the original organization, subsequent growth, and present plans of the AMERICAN FEDERATION FOR CLINICAL RESEARCH.

#### THE BASIC PLAN

Thirteen surviving charter members of the American Society for the Advancement of Clinical Investigation met with Henry Christian, on May 6, 1940, to plan a new society to supplement the present American Society for Clinical Investigation. The purpose of this new society would be "to stimulate among young men a persisting interest in investigation in clinical and allied medical sciences." In November of that year the idea and the constitution were proposed, by letter, to interested persons. This letter was signed by the fourteen mentioned above, including such outstanding men as Capps, Christian, Emerson, Libman, Longcope and Pratt. The first meeting in Atlantic City, May 5, 1941, was attended by 300 young men and women, who approved the constitution and by-laws presented by Dr. Christian. These original suggestions stressed that membership would be open to any young man or woman who was interested in and was doing research in any branch of science dealing with the application of the healing art, such as medicine, surgery, obstetrics, gynecology, pediatrics, pathology, bacteriology and chemistry. It was hoped that such persons would be welcomed into our membership even if they were not connected with medical schools or research institutes. Membership was

intended primarily for persons under 35 years of age, and no person was to hold office after the age of 40. There was to be no numerical limitation to the Society, but a prospective member was to be required to present tangible evidence of interest in clinical investigation in the form of at least one completed piece of research. A single case report was not to be considered sufficient accomplishment to earn membership. Each prospective member was to be judged by what he was doing and had done, rather than by letters of recommendation.

Another feature of the Society was the organization of local research clubs. "Such clubs may be organized in any community by any interested group of investigators. The jurisdiction of such a club lies with itself and is not under the AMERICAN FEDERATION. But it is hoped that members of the AMERICAN FEDERATION will make themselves responsible for the organization of such 'research clubs' and to be the sustaining force."

Dues were to be low, in keeping with the meager income of the young members. For the same reason, no member was to be required to attend every national meeting. Also, since National Councilors were chosen from the same financially limited members, the Society was to be responsible for at least a part of the councilors' traveling expenses to national meetings, whenever necessary.

In 1948 it was further urged "that expenses of the Sectional meetings are to be kept as low as possible . . . that the budget allowed by the National organization for Sectional meetings should not include any refreshments. Insofar as possible, lantern slides and operators are to be furnished by members of the organization, and whenever feasible, such meetings are to be held in places where no charge will be made for the use of an auditorium. In summary, the national organization will underwrite only such expenses as are absolutely necessary for the holding of Sectional meetings."

#### GROWTH AND CHANGE

The rapid growth of local, regional, sectional and national meetings best demonstrated the need for, and at the same time promoted the principal endeavor of, our society to create a welcome place

\* Presidential Address, read before the National Meeting of the American Federation for Clinical Research, Atlantic City, New Jersey, May 1, 1955.

for every competent beginner in medical research to present the results of his work. This growth has been paralleled by an increase in total membership to nearly 2500.

Yearly national meetings have been held ever since that original meeting in 1941, with the exception of 1943, when the meeting had to be canceled because of World War II restrictions on travel. The first sectional meeting was held in the Midwest, November 4, 1943. This was followed in rapid succession by Southern, Eastern and Western sectional meetings. The first regional meeting was held in Michigan on March 10, 1945. Regional and local meetings have been held in more than 300 locations. The financial association of these meetings with the National organization has been outlined above. Further liaison was developed by urging local and regional chairmen to report activities in their areas to the chairman of their annual sectional meeting. The sectional chairmen, in turn, send a copy of their revised rosters of local membership, minutes of local meetings and programs to the office of the National Secretary following each sectional meeting.

Some of the first meetings were held in small university halls, where the speaker realistically addressed his audience as "fellow essayists!" This unpromising start gave way to crowded hotel ball-rooms, and finally, in 1953, the national meeting grew to fill the large auditorium of the Steel Pier in Atlantic City.

The AMERICAN FEDERATION saw to it that the beginner in research not only was heard, but also that abstracts of the results of his work could be read while the work was still timely. Eli Lilly voluntarily published the first PROCEEDINGS in 1944. Subsequently, almost every pharmaceutical house in the United States generously helped defray publication expenses during our early phase of deficit financing. The Society started publishing its abstracts in *The American Journal of Medicine* in 1947. Growth of membership and productivity of members soon required the Society to negotiate for a journal of its own. In 1953, Grune & Stratton published the first issue of CLINICAL RESEARCH PROCEEDINGS, with Dr. Lawrence E. Hinkle, Jr., as editor pro tem. Dr. David Graham was appointed editor in May 1954.

#### 1955—?

Where do we go from here? Several innovations were made during this past year. In an attempt to

strengthen the AMERICAN FEDERATION at its roots, the National Office sent a notebook containing a formal charter to each local or regional group. This book also contains ground rules outlining the relationship between local groups and the National Organization. The charter and ground rules were suggested by councillors and sectional chairmen in an effort to give aid and continuity to local meetings. We have increased the frequency of publication of CLINICAL RESEARCH PROCEEDINGS in order to insure more prompt publication of our abstracts. CLINICAL RESEARCH PROCEEDINGS has become more than an abstract journal, however, and now experiments by presenting thoughts about research and instructive notes for young investigators. Our organization has raised dues only when it felt certain that inflation was here to stay and our new journal had to be financed. In order to stimulate further interest in investigation and to give more young men a place to be heard, we will experiment this evening by holding three informal subsections on Cardiovascular Disease, Renal and Electrolyte Metabolism and Medical Education. Lastly, we have notified the American Society for Clinical Investigation and the Association of American Physicians that we welcome a change in the geographic location of our national meetings, so that the two-thirds of our membership who live at some distance from the eastern coast will not be consistently inconvenienced by unnecessary travel.

The AMERICAN FEDERATION FOR CLINICAL RESEARCH might well feel proud to look back at the aims proposed in the original organization in 1941 and note that, in spite of the stresses and strains of rapid growth, a constantly moving membership and a shoestring budget, the latest Constitution shows furtherance of the original plan. The chance for a beginner in medical research to be heard and read has been promoted with increasing success. The history of similar organizations, however, demonstrates that the task of future officers will become increasingly difficult in promoting the original aims of giving "a place to young men, beginners in investigation, and to men not connected with medical schools or research institutes . . ." and "to stimulate among young men a persisting interest in investigation in clinical and allied medical sciences. . . ." Let us constantly struggle to maintain the vitality of our endeavor to keep a welcome place.



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## Lilliput Revisited\*

By Edward M. Schneider†

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FOR MANY YEARS medical scientists have been wrestling with the riddle of the mechanism of pain in peptic ulcer. Most gastroenterologists have come to accept either hyperacidity or intestinal dyskinesia as the basic mechanism. The crux of the problem lies not so much with the definition of "hyperacidity" or "intestinal dyskinesia" as it does with the use of the smaller and simpler word "either." The latter word implies mutual exclusion and hence virtual irreconcilability of the two possibilities. Indeed, the heated discussions and the only slightly less warm publications that have appeared in the literature from time to time are reminiscent of Blefuscu and Lilliput and their bloody battles over the proper end at which to open a soft-boiled egg.

For both theories, evidence of two types can be presented: one that positively points to the particular theory's accuracy, and the other that inferentially supports one view by failure to support the other.

In support of the intestinal dyskinesia theory, positive evidence, as recently presented by Bloomfield,<sup>1</sup> consists of: the observation that inflation of a small balloon in the duodenum may reproduce the pain of duodenal ulcer although the only apparent measurable physiologic effect of the procedure is increased motility; the report of gastroscopists who associate violent contractions of the pylorus with the onset of pain and relief as spasm subsides; the recent findings of Ruffin et al.<sup>2</sup> that an acid-barium mixture in an ulcer crater might not be associated with pain unless "alterations in normal motility" were also seen.

Other evidence, also cited by Bloomfield,<sup>1</sup> has been presented as opposing the acid theory, and hence supporting the intestinal dyskinesia theory: appearance of an ulcer without symptoms, that may go on to perforation; appearance of ulcer symptoms in patients who have no gastrointestinal disease whatsoever; persistence of characteristic ulcer pain in patients whose lesion has been removed surgically; the "hyperacidity" symptoms that sometimes occur in patients with hypochlorhydria or achlorhydria; relief of pain by sedation when there is no apparent

change in degree of gastric acidity; relief of pain by doses of alkali too small to neutralize the acid present.

On the other side of the question, the arguments rely on the following evidence.\* Kymographic and x-ray studies of the stomach and duodenum fail to reflect a coordination between hyperperistalsis, gastric contraction and ulcer pain; ulcer distress occurs only when gastric content is acid, although the degree of acidity can vary from patient to patient; typical ulcer pain can be produced by the instillation of physiologic concentrations of hydrochloric acid in the stomach of a patient with peptic ulcer; typical pain can be relieved by aspiration, neutralization or drug inhibition of acid gastric secretions.

As more oblique support for the hyperacidity theory, it has been suggested: that motor activity can be increased by strong acid, and thus lead to the development of pain<sup>4</sup>; that, if spasm is the cause of peptic ulcer pain, it would be reasonable to assume that all human beings would have frequent bouts of typical ulcer pain, since spasm or hypermotility must occur in the process of normal digestion. The fact that this does not occur has been taken as evidence that intestinal hypermotility is not the cause of pain.<sup>1</sup> It has been further noted that the usual ulcer pain is of a burning or gnawing character—a type of discomfort associated with acid in most open lesions, whereas hypermotility or spasm produces a cramp-like discomfort.

It is not the intention of this article to refute, support or elaborate on any of the arguments cited. These statements have all been made in the literature within the past three years. The obvious reconciliation of the conflicting views lies in the inference that any adequate stimulus is capable of eliciting pain from a suitably susceptible mucosa, whether it be "burned" by acid or distorted through spasm. The pain of peptic ulcer is probably not the exclusive province of *either* hyperacidity or hypermotility. There is evidence that associated hyperemia and engorgement of the mucosa of the stomach and intestinal tract may so lower the pain threshold that ordinarily inadequate stimuli may be adequate to produce pain.<sup>6</sup> Pain therefore seems to depend upon gastric and duodenal hyperfunction in general. Any aspect—hyperacidity, hypermotility or mucosal engorgement, or any combination of these factors—may be predominant.

The egg will open, regardless of where it is cracked.

\* The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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## Host Factors and the Decline of Infection

By Ivan L. Bennett, Jr.\*

AT RECENT MEDICAL GATHERINGS (such as the meetings of the American Federation for Clinical Research) it has been common to hear statements to the effect that infectious disease is dying on the vine, or that the bug-doctors are going downhill. Most "infectious disease men" are not overly sensitive and are willing to accept these expressions of disenchantment at somewhat less than face value (occasionally offering in rebuttal a few remarks about the blood volume in congestive heart failure, life-span of erythrocytes, tubular maximums,  $I^{131}$  uptakes, or life situations, stress, etc.). However this may be, a change of emphasis is occurring in the experimental study of infection. Superficial interpretation of this change might account for the idea that infection as a subspecialty of internal medicine is becoming anachronistic. As is often the case, this change is not really new; it is a renewal of interest in what Dubos has recently called "non-microbial determinants of infectious disease," in the examination and exploration of nonspecific factors (many of them as well known to the laity as to the profession) which influence host susceptibility and host resistance.

Before discussing some of the reasons for revival of interest in this area, one further preliminary statement is in order. By and large, microbiology has outgrown the stage which Schwartzman terms the "era of specificity." Discovery of specific causative organisms of disease (particularly bacteria) no longer occupies the center of the stage. A notable exception to this is the tremendous advance in virology, as new technics of cultivation (currently tissue culture) are applied on a wider scale. It has become a relatively easy matter to discover new viruses. To find diseases to assign them to is increasingly difficult. The classification of new agents

and the determination of their epidemiologic and immunologic spectra are highly specialized and singularly tedious technical undertakings, the details of which are seldom imposed upon those attending general medical meetings. Rather, items of more clinical interest such as the relationship between Cocksackie viruses and Bornholm disease, or between Hilleman's ARD virus or Huebner's APC viruses and common respiratory disease seem appropriate subjects for most gatherings of clinicians and clinical investigators; the detailed experimental procedures are usually reserved for more specialized groups and symposia. While a tabulation of signs, symptoms, prognosis and attempts at therapy of epidemic hemorrhagic fever, or even a discussion of renal hemodynamics and changes in blood volume at various stages of this disease, would be heard with at least mild interest by many, the same cannot be said for a paper outlining the various microbiologic, immunologic and epidemiologic manipulations that have been used in attempts to isolate its causative agent.

To return to "nonspecific" factors governing infection, there are at least four origins of the present resurgence of interest in host susceptibility and host resistance. The first of these, which will be mentioned briefly, relates to many observations upon species and genetic variations in susceptibility. Why are mice resistant to infection with Type XIV pneumococcus? What factors are responsible for the host range of the avian, bovine and human types of tubercle bacilli? In what way do the rabbits bred by Lurie for resistance to tuberculosis differ from their susceptible cousins? Why does inbreeding of laboratory animals for resistance to one infection often lead to concomitant increased susceptibility to other infectious agents? There are a few instances in which leukocyte count, body temperature, or perhaps adrenal function seem to account for species or strain resistance to infection, but in most cases,

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although the bare fact of susceptibility or insusceptibility is easily established, nothing more is known.

A second source of interest in this question may be termed epidemiologic. It has long been known and accepted that the mere presence of pathogenic bacteria or viruses in the tissues of a host does not necessarily result in symptoms of illness, and such terms as "latent," "subclinical," and "inapparent" infection or "asymptomatic carrier" are employed frequently. Of countless persons who harbor meningococci in the nasopharynx each winter, only a few develop meningococcal disease. While many possible explanations for the sudden invasion that occurs in these few unfortunates can be suggested, the truth of the matter is that we simply do not know why this subclinical infection becomes clinical disease. Chilling, fatigue, concomitant upper respiratory infection, specific antibodies, malnutrition, etc., cannot, as yet, be shown to bear any relationship to this important transition. The example of latent infection with herpes simplex virus is well known. In virtually all adults this symbiont lies dormant, until, under various kinds of stress—some obvious, others obscure—the appearance of a crop of vesicles signifies that even in the presence of specific antibody the virus has again produced disease. What chain of events do these stimuli initiate that enables the breakthrough to occur? Why is labial herpes rare in typhoid fever and yet almost routine in patients given typhoid vaccine for fever therapy? Why are fever blisters frequent in malaria, pneumococcal and meningococcal infections, influenza, scarlet fever, typhus and relapsing fever, and rare in tuberculosis, smallpox and brucellosis? Why do patients recovered from typhoid sometimes continue to harbor virulent bacilli for years? Recovery from this disease is not demonstrably a function of humoral antibody; illness often continues for long periods after antibodies appear, and relapses can occur in the presence of high levels of specific immune bodies. Why is not the typhoid carrier as sick as his contacts? One need only mention "abortive" poliomyelitis and serum hepatitis, two among many other striking instances where the presence of the specific causative agent of a disease does not, in the absence (or presence) of some other factor, mean the occurrence of recognizable disease.

Intriguing as questions raised by species and genetic variations in resistance and by inapparent infection have been for so many years, the current renewal of interest in factors other than the presence of a microorganism which influence infectious disease stems more directly from the advent of specific antimicrobial therapy. Besides offering effective treatment for many diseases, sulfonamides and antibiotics have gradually brought into sharp focus the importance of host participation in infection, and in recovery from the infectious process. The practical usefulness of these drugs has not

eliminated but has made more important than ever the need for basic understanding of the mechanisms underlying and influencing the onset and subsidence of infection. It is relatively easy to understand the beneficial effects of bactericidal drugs such as penicillin, streptomycin or bacitracin. If the infecting organisms are killed, one would certainly expect recovery unless some lethal or irreversible damage had already occurred. However, the sulfonamides, the tetracyclines and chloramphenicol are primarily bacteriostatic agents, able simply to prevent multiplication of bacteria, not to kill them directly. Administration of one of these suppressive agents for an adequate period can bring about complete cure of infection (not only clinically but also bacteriologically). In other words, if bacterial multiplication is inhibited for a long enough time, organisms disappear completely. This implies, of course, that final eradication of microbes is dependent upon the infected host's making a contribution in his own defense. The studies of Wood on the mechanism of recovery in pneumococcal pneumonia have certainly demonstrated the importance of phagocytosis as a supplement to drug-induced bacteriostasis. A better understanding of some of the other factors which enable the tissues of the host to "join forces" with administered antibiotics would unquestionably be reflected in better clinical results in using these agents. Secondly, antibiotics have made it clear that, in certain types of infection, the specific microbial agent is of considerably less importance than is the anatomic site of the infection. The pneumococcus is sensitive to small amounts of penicillin and has not been observed to develop significant resistance to penicillin. Pneumococcal pneumonia is easily cured with a few hundred thousand units of penicillin. But when the pneumococcus produces infection in the meninges, many millions of units of penicillin daily may fail to eliminate infection; the *pneumococcus* is still "sensitive" to penicillin but the infection is now relatively insensitive to it. Some of this may be due to poor diffusion of the drug into certain sites, impaired function of phagocytes in the meninges, etc., but the mechanisms responsible for the empiric fact that one type of infection requires small amounts of drug for a short time, while another type requires large amounts for a long time, are poorly understood. Bacterial endocarditis is another disease in which "sensitive" organisms can produce "resistant" disease. Only by continued attempts to elucidate the physiologic and biochemical factors operative in the diseased tissues of the host can the eventual solution to these problems be reached.

Lastly, impetus has been given to the study of host factors in infection by the increasing availability of methods for altering resistance. Adrenal steroids, nitrogen mustard, ionizing radiation, Urethane, various dietary regimes, even so simple a procedure as administration of citrate, or indeed, of

glucose, have been employed successfully to increase susceptibility; in no instance is the mechanism understood, although it is clear that the change is in the host and not the microorganism. Experimentally, certain other procedures such as obstruction of lymphatics (or any hollow tube such as a ureter or bronchus), creation of A-V fistulas, exposure in a high altitude chamber, or inoculation of organisms in mucin have been employed to increase the incidence of spontaneous infections or enhance the severity of induced infections. None of these is clearly understood, and many are under intensive study; but it may well be that only when we understand the "normal" course of infection will we be able to define the significant alterations brought about by such manipulations. Even the administration of an antibiotic can influence resistance to the detriment of the host. The striking experiments of

Miller on *Salmonella* infection in mice after alteration of intestinal flora by antibiotics illustrate this so-called "superinfection."

In summary, I believe that a partial explanation for the apparent "decline of infection" at clinical research meetings is to be found in the fact that renewed interest in the *host* has forced the infectious disease man to turn from the time-honored technics of microbiology, and to borrow from surgery, biochemistry, cytology, physiology, nutrition, etc. Studies on the carbohydrate metabolism of polymorphonuclear leukocytes, the preparation of spermine in crystalline form or chromatographic separation of adrenal steroids are as important as the development of new technics for determining bacterial sensitivity to antibiotics—all are now within the realm of the infectious disease man.

### Erratum

The Publisher regrets the appearance of a typographic error in Dr. Albert I. Mendeloff's article in the April 1955 issue of CLINICAL RESEARCH PROCEEDINGS. On page 86, first paragraph of the right-hand column, it was stated:

"What will happen to one and a half million irradiated stomachs during a forty-five year span? Other chronic diseases have not been shown to begin many years before their symptomatic manifestations: duodenal ulcer is a disease of the twenties, gastric ulcer of the late thirties—must one seek their origins in childhood or adolescence?"

The word **not** should have read **now**.

# NOTICES

## Forthcoming Meetings

**Midwestern Section:** The Annual Meeting will be held on Thursday, November 3, 1955 at 9:00 a.m. in the auditorium of Thorne Hall, Northwestern University Medical Campus, Lake Shore Drive and Superior Street, Chicago, Illinois. Headquarters will be at the Drake Hotel. Abstracts (see rules below) must be postmarked not later than midnight August 31, 1955 and sent to Dr. Robert J. Glaser, Barnes Hospital, 600 South Kingshighway, St. Louis 10, Missouri.

**Eastern Section:** The Annual Meeting will be held on Friday, December 2, at 9:00 a.m. and on Saturday, December 3, 1955 from 9:00 a.m. until noon in the College of Physicians Building, 19 South 22nd Street, Philadelphia, Pennsylvania. Headquarters will be at the Hotel Warwick. Abstracts (see rules below) must be postmarked not later than midnight October 1, 1955 and sent to Dr. Charles R. Shuman, Temple University Hospital, Broad and Ontario Streets, Philadelphia 40, Pennsylvania.

**Southern Section:** The Annual Meeting will be held on Friday, January 20, 1956 at 9:00 a.m. in the Jung Hotel, New Orleans, Louisiana. Abstracts (see rules below) must be postmarked not later than midnight November 15, 1955 and sent to Dr. John H. Moyer, Baylor University College of Medicine, 1200 M. D. Anderson Boulevard, Houston, Texas.

**Western Section:** The Annual Meeting will be held on Wednesday, January 25, 1956 at 9:00 a.m. in the Golden Bough Theater, Carmel, California. Headquarters will be at the La Playa Hotel. Abstracts (see rules below) must be postmarked not later than midnight November 15, 1955 and sent to Dr. B. H. Scribner, Veterans Administration Hospital, Seattle 8, Washington. (Note: The call for abstracts listed the Western Section Meeting January 25, 1956. This has been changed, as shown above, to January 26.)

## Rules for Preparation of Abstracts

(1) Submit 5 copies. This will permit the Section Council to aid in program selection.

(2) Abstracts must not exceed 300 words and must be double-spaced.

(3) Authors should follow the form outlined in *CLINICAL RESEARCH PROCEEDINGS* 1: 120, September 1953.

(4) Abstracts may also be submitted to the parent Society with which the Section meets. The Section Chairman should be informed, if this is done.

(5) One of the authors must be a member or the paper must be introduced by a member. The one who reads the paper must not be over 40.

(6) Membership status of authors should be included to facilitate preparation of the printed program.

## Applications for Membership

Anyone who wishes to become a member of the American Federation for Clinical Research should write to the National Office (Dr. William W. Stead, Secretary-Treasurer, Veterans Administration Hospital, Minneapolis 17, Minnesota) or get in touch with the geographic Section of his local group. The annual subscription to *CLINICAL RESEARCH PROCEEDINGS* is \$3.00 for members and is included in the \$5.00 annual membership dues. Remittances should not be included with applications for membership.

Membership applications will be voted on at intervals throughout the year, and thus eliminate the former long delay.

## Obituaries

The National Office has been notified of the death of the following members:

Dr. Ellery G. Allen, Syracuse, New York, on January 4, 1955.

Dr. Leon Levinson, Boston, Massachusetts, in January, 1955.

Dr. Thomas H. Lorenz, University Hospitals, Madison, Wisconsin, on June 18, 1955.

Dr. Ferdinand R. Schemm, Great Falls Clinic, Great Falls, Montana, on May 16, 1955.

Dr. John R. West, Columbia University, in June, 1954.

## Members Lost to the National Office

It would be appreciated if each member would review the following list and notify Dr. William W. Stead of the current address of anyone on the list whom he recognizes:

Richard C. Bentinck  
Max W. Biggs  
W. J. Butt  
Harold O. Conn  
Roy C. Crosby  
Simon Dack  
William G. Dixon  
Oscar Felsenfeld  
John J. Finn  
Vincent J. Fontana  
George L. Forbes, Jr.  
Goffredo G. Gensini  
Edwin E. Goldberg  
Irwin B. Hanenson  
Marvin M. Hirsch  
Erwin Huston

Robert L. Johnson  
Edwin C. Jungck  
Robert J. Kalthoff  
Ceylon S. Lewis, Jr.  
David Littman  
Meyer Markovitz  
Albert G. Marrangoni  
Theodore B. Massell  
Stanley Newman  
Robert O. Oseasohn  
Paul Pfeiffer  
Gerald I. Plitman  
Walter C. Ralston  
Donald W. Rennie  
Robert B. Rutherford



Mary L. Scholl  
John R. Sheehan  
Santos Silva  
Franz R. Stenzel  
Virginia M. Stuermer  
Marguerite P. Sykes

Robert D. Taylor  
Melvin L. Taymor  
Joseph B. Trainer  
Bernard M. Wagner  
Mackenzie Walser  
Walter S. Wiggins

### Reprints

Contributors to CLINICAL RESEARCH PROCEEDINGS will find information regarding reprints of abstracts and articles on the masthead page (iv) of this and all succeeding issues of the Journal.

### Supply of Rhesus Monkeys

As result of negotiations, the United States Mission to India and the United States Embassy in India have secured the agreement of the Government of India to permit the continued exportation of rhesus monkeys for medical research and production of antipoliomyelitis vaccine. All needs must be

certified by the United States Government through the Public Health Service.

Information concerning Public Health Service procedure and Certificate of Need forms may be secured from: National Institutes of Health, Room 1012, Building T-6, Bethesda 14, Maryland (Telephone: Oliver 6-4000, Extension 2748, Miss Parent).

### Films

The Academy-International of Medicine, 601 Louisiana Street, Lawrence, Kansas, announces that a completely revised fourth edition of *Professional Films* is now in compilation. It will include new sections providing biographic data on authors, and information on the audiovisual activities of medical schools, dental schools and postgraduate teaching centers. Interested film authors may write for information.

# 1955 Membership Roster

## of the

### American Federation for Clinical Research

No Asterisk—Full Member

\* Associate Member

\*\* Senior Member

- Frank K. Abbot, 3700th USAF Hosp., Lackland AFB, San Antonio, Tex.
- Ossler A. Abbott,\*\* Emory University Hosp., Emory University, Ga.
- William E. Abbott,\*\* University Hosp., 2065 Adelbert Rd., Cleveland, O.
- Stuart Abel, 700 Michigan Ave., Chicago 11, Ill.
- Walter H. Abelmann, Heart Station, Boston City Hosp., Boston 18, Mass.
- J. A. Abildskov, Box 44, William Beaumont Army Hosp., El Paso, Tex.
- Joseph P. Abraham, Henry Ford Hosp., Detroit 2, Mich.
- Edward W. Abrames,\*\* Paulsen Medical & Dental Bldg., Spokane, Wash.
- William B. Abrams, Apt. 26, 466 South Center St., Orange, N. J.
- Benedict E. Abreu,\*\* Pitman-Moore Co., 1200 Madison Ave., Indianapolis 6, Ind.
- Charles V. Adair, 1010 Woodland Rd., Mansfield, O.
- Thomas W. Adams,\* 11 Parkway Dr., Edgewood Hills, East Peoria, Ill.
- Richard H. Adler, Dept. Research & Development, Fitzsimons Army Hosp., Denver 8, Colo.
- David Adlersberg,\*\* Mount Sinai Hosp., New York 29, N. Y.
- Paul M. Aggeler,\*\* 655 Sutter St., San Francisco 2, Calif.
- Elbert B. Agnor,\*\* 1211 W. Peachtree St. N.E., Atlanta, Ga.
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- Howard E. Allen,\*\* 1216 S.W. Yamhill St., Portland, Ore.
- H. R. Allumbaugh,\*\* Medical Center, Eugene, Ore.
- Thomas P. Almy, 2nd Medical Division, Bellevue Hosp., New York 16, N. Y.
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- Charles H. Altshuler, St. Joseph's Hosp., Milwaukee, Wis.
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- Thomas Tarcisio Amatruda, Jr., Grace-New Haven Community Hosp., New Haven, Conn.
- William H. Ames, 902 Edmond St., St. Joseph, Mo.
- Ellsworth L. Amidon,\*\* 144 De Forest Rd., Burlington, Vt.
- Basil D. Anagnost,\*\* 123-17 St., Toledo 2, O.
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